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Review article

Congenital coronary anomalies

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ABSTRACT

Background: Congenital coronary anomalies occur rarely and affect 0.3–1% of the population [1,2]. They are predominantly clinically silent and are usually diagnosed accidentally during selective coronarography for other reasons, or at autopsy. In some cases however these may be the cause of sudden death, mainly in young individuals in association with great exertion (anomalous origin of coronary artery from the opposite coronary sinus with interarterial course).

Method: We reviewed the cathlab records and coronary multi-slice CT scans performed in the period from 2008 to 2013 in our hospital and we chose typical image findings documenting particular congenital coronary anomalies. Subsequently we reviewed the literature using the PubMed database.

Results: In our image summary, we discuss these particular anomalies and primarily their imaging documentation, using either multi-slice CT or selective coronarography, which represents the main diagnostic methods for these disorders.

Conclusion: Congenital coronary anomalies are relatively rare findings; however, some of these could have significant and even fatal consequences. These disorders should be considered in selected risk population groups. Multi-slice CT coronarography seems to be the most appropriate diagnostic method with regard to the possibility of 3D imaging.

Contents

Abbreviations: LM/LMCA, left main coronary artery; LAD, left anterior descending artery; LCx, left circumflex artery; RCA, right coronary artery; CS, coronary sinus; Ao, aorta; Pu, pulmonary artery; LA, left atrium; RA, right atrium

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Introduction

Congenital coronary anomalies (CAA) occur rarely and affect 0.3–1% of the population [1,2]. In 87% of cases these anomalies are in the origin and course of coronary arteries and in 13% of cases they include fistulas [3]. These anomalies are predominantly clinically silent and are usually diagnosed accidentally during selective coronarography for other reasons, or at autopsy. In some cases however these may be the cause of sudden death, mainly in young individuals in association with great exertion (ACAOS with interarterial course). CAA could be the cause of sudden death in 5–35% of cases in young athletes or recruiters [4–7]. On the contrary, they are the cause of only 0.6–1.2% of all sudden deaths in young patients [6,8]; hence, they are fatal only in association with or shortly after extreme physical exertion.

Pathophysiology and clinical symptoms

Embryonic development of coronary arteries is a complex process that starts on the surface of the heart muscle, which only subsequently leads to the development of their connection to the aorta. This may explain the variability of coronary supply and development of anomalous central connection with the aorta [9]. The extent of clinical symptoms depends on the type and haemodynamic significance of congenital coronary anomalies and includes clinically completely silent course (in most of the anomalies) up to sudden death, mainly in young individuals in association with extreme exertion. Other symptoms may be variable and include atypical chest pain, shortness of breath, arrhythmia, development of heart failure or acute myocardial infarction. CAA however only rarely causes reproducible angina pectoris at a certain level of exertion that is otherwise typical for ischemic heart disease [2]. Although there is no definite opinion regarding the relation between CAA and coronary atherosclerosis, according to the predominant opinion, the anomalous segments are not more prone to atherosclerosis [2]. CAA could occur separately or could be associated with other congenital heart disorders.

Diagnostics

Since congenital coronary anomalies often manifest only during extreme exertion, classical submaximal exercise tests (exercise ECG test or Tc SPECT) fail during their diagnostics [2]. The oldest diagnostic method is selective coronarography. This is however an invasive method and due to 2D visualization it is often difficult to evaluate the course of anomalous coronary arteries with regard to the other heart structures and large vessels. Selective cannulation of atypical origins of coronary arteries may also be technically difficult. Echocardiography is a non-invasive method that enables visualization of the origins and proximal segments of coronary arteries; however, a suitable trans-thoracic echocardiography window is available only in children or adolescents. Transoesophageal echocardiography is already a semi-invasive method. MRI would be an ideal non-invasive low-demanding examination method; its spatial resolution is however insufficient. The most appropriate imaging method is currently multi-slice CT coronarography (MS-CT), which has a sufficient resolution and enables 3D imaging evaluation of the course of coronary arteries in relation to the large vessels and other heart structures. The disadvantage is the radiation burden.

Classification

Normal anatomy

Coronary arteries normally originate under the sinotubular junction from the middle of the corresponding coronary

sinus. The right coronary artery (RCA) originates from the right coronary sinus and the left coronary artery (LCA) from the left one, which branches after a short course (the left main stem – LMCA) to the left anterior descending (LAD) and left circumflex coronary artery (LCx). In some cases there could be a separate origin of the LAD and LCx.

Modified classification of anomalies according to Greenberg: [10]

Anomalies of origin:
- High takeoff
- Multiple coronary ostia
- Single coronary artery
- Anomalous origin of the coronary artery from the pulmonary artery
- Anomalous origin of coronary artery from the opposite and non-coronary sinus (ACAOS)

Anomalies of course:
- Myocardial bridging
- Duplication of arteries

Anomalies of termination:
- Coronary artery fistula
- Coronary arcade
- Extra-cardiac termination

Coronary anomalies can also be classified as hemodynamically significant or insignificant. Haemodynamically significant anomalies can cause impaired myocardial perfusion and result in myocardial ischemia, heart failure, or sudden cardiac death.

Anomalies of origin

High takeoff

This means high takeoff of RCA or LAD from the ascending aorta, most often 1 cm above the sinotubular junction. In some cases it could also originate more distally, from the area of the arch or its main branches. These anomalies are usually hemodynamically insignificant, and may only cause technical problems during selective coronarography. In some cases the artery could have an intramural course in the aorta wall and could be compressed during exertion [9]. (Fig. 1)

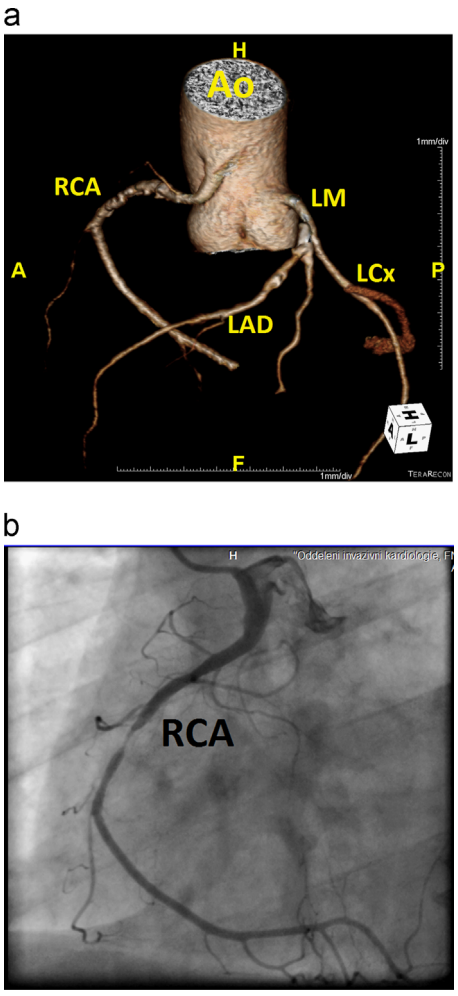

Fig. 1 – The right coronary artery (RCA) high takeoff. a – MS-CT volume-rendered 3-D image; b – selective coronarography: left anterior oblique 45° view, a tight stenosis of middle segment of RCA.

Multiple coronary ostia

Most often the RCA and conus branch arise separately from the right coronary sinus or the LAD and LCx arise separately from the left coronary sinus, without LM. Furthermore, 1 septal branch can originate directly from the aorta. These findings are hemodynamically completely insignificant. With regard to their frequent occurrence (in up to 30–50% of patients) they could be considered a normal variant [9].

Single coronary artery

This is a very rare anomaly, which occurs only in 0.0024–0.044% of cases [1], when only 1 coronary artery arises from the aorta, which may have its further course similar to normal LCA and RCA, or it may be completely anomalous. In 41% of cases it is associated with other congenital heart diseases (such as tetralogy of Fallot, transposition of large vessels, etc.) [9]. If its course is intra-arterial between the aorta and pulmonary artery, these patients are at a high risk of sudden cardiac death; furthermore, stenosis or thrombotic occlusion of the proximal segment of this artery may also have fatal consequences.

Anomalous origin of coronary artery from the opposite or non-coronary sinus (ACAOS)

Coronary arteries could arise from the opposite or non-coronary sinus. The actual anomalous origin of coronary arteries is not a problem, but its further course, by which the coronary artery reaches its perfusion area, could have severe clinical consequences. The anomalous coronary artery can take any of the following four common courses, depending on the anatomic relationship of the anomalous vessel to the aorta and the pulmonary trunk:

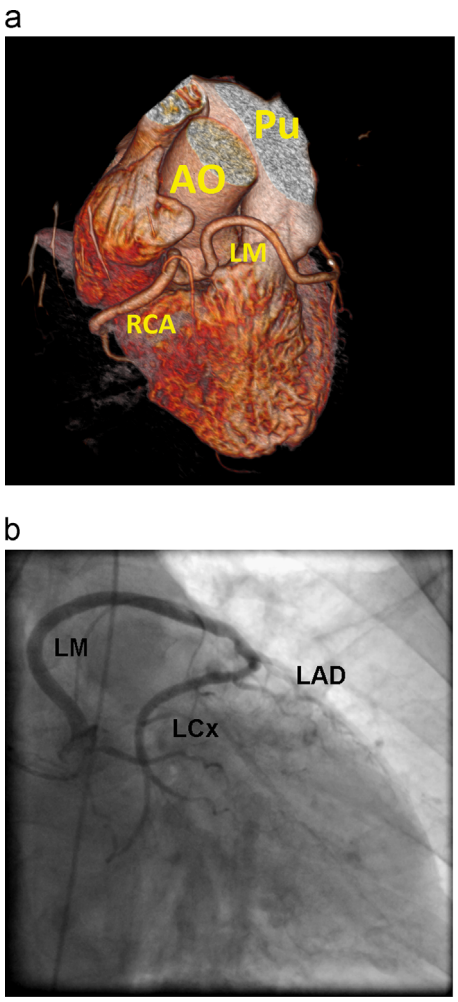

Fig. 2 – The left main coronary artery (LM) arising from the right coronary sinus with prepulmonic course. a – MS-CT volume-rendered 3-D image; b – selective coronarography: right anterior oblique 30° caudal 20° view.

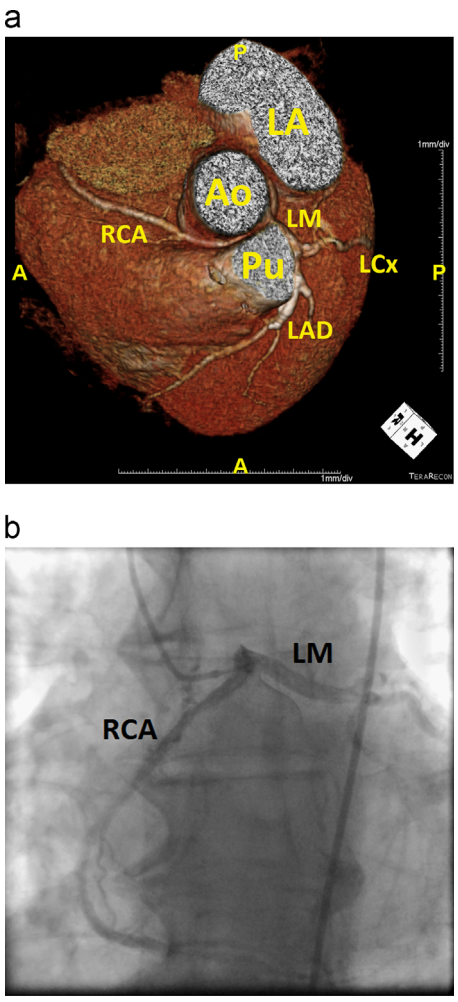

Fig. 3 – The right coronary artery (RCA) arising from the left coronary sinus with interarterial course. a – MS-CT volume-rendered 3-D image; b – selective coronarography: postero-anterior view.

prepulmonic (Fig. 2)
interarterial – between the aorta and the pulmonary trunk (Figs. 3 and 4)
intraseptal or
retroaortic course (Figs. 5 and 6)

While the prepulmonic, intraseptal and retroaortic courses are considered to be benign, the interarterial course is often associated with an increased risk of sudden cardiac death. This course could represent up to a 30% risk of sudden cardiac death and it is reported as a cause of sudden death in 5–35% of young individuals in association with intensive exertion [4]. Compared to this, the risk of sudden death in middle-aged and elderly individuals with ACAOS is usually low. ACAOS could be associated in up to 26% of cases with some other aortal root anomaly, e.g. with the bicuspidal aortal valve [11]. The RCA arises separately from the left coronary sinus or as a branch of the LCA in 0.03–0.17% of patients [1]. Most frequently, in up to 90% of cases [9] there is an interarterial course, and this could be associated with the risk of sudden cardiac death in up to 30% of cases [1]. The anomalous origin of LMCA from the right coronary sinus or as a branch of the RCA is found in 0.09–0.11% of patients [1]. In 75% of cases there could be an interarterial course associated with a high risk of sudden cardiac death [1]. Anomalous LCA could however also take prepulmonic, intraseptal or retroaortic course. LCx or LAD could also anomalously arise separately from the right coronary sinus. LCx often has a retroaortal course [1]. Anomalous origin of LAD is mostly associated with other congenital heart disorders (such as tetralogy of Fallot, transposition of large vessels, etc.); it is only rarely isolated. It could have an interarterial as well as prepulmonary course [1]. LCA or RCA could also arise from the non-coronary sinus; however, these anomalies are rare in an otherwise normal heart, they are often associated with the transposition of large vessels [1].

The definite pathophysiological mechanism of sudden cardiac death is not clear. A traditional hypothesis explaining the cause of sudden cardiac death in ACAOS with an interarterial course often reported a slit-like ostium and sharp angle (below 45°), under which the anomalous artery arises from the opposite sinus, which could create a valve-like ostial

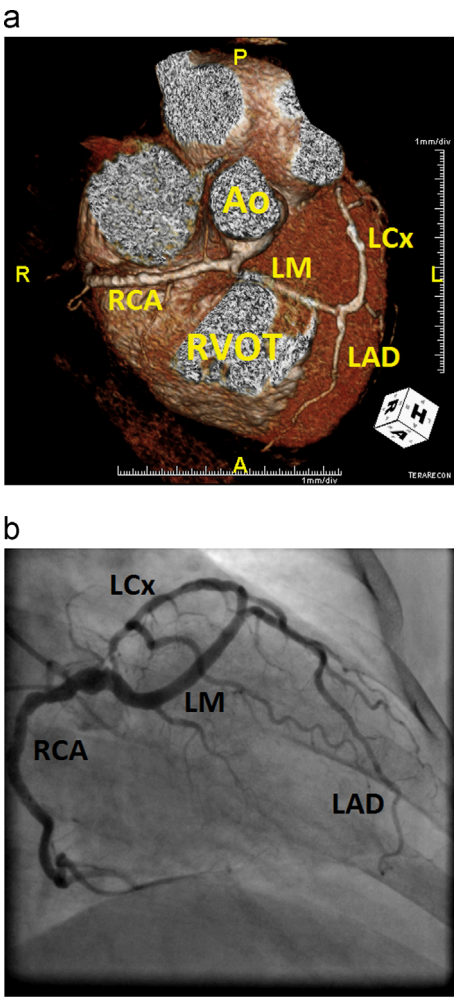

Fig. 4 – The left main coronary artery (LM) arising from the right coronary sinus with interarterial course. a – MS-CT volume-rendered 3-D image; b – selective coronarography: right anterior oblique 15° caudal 15° view.

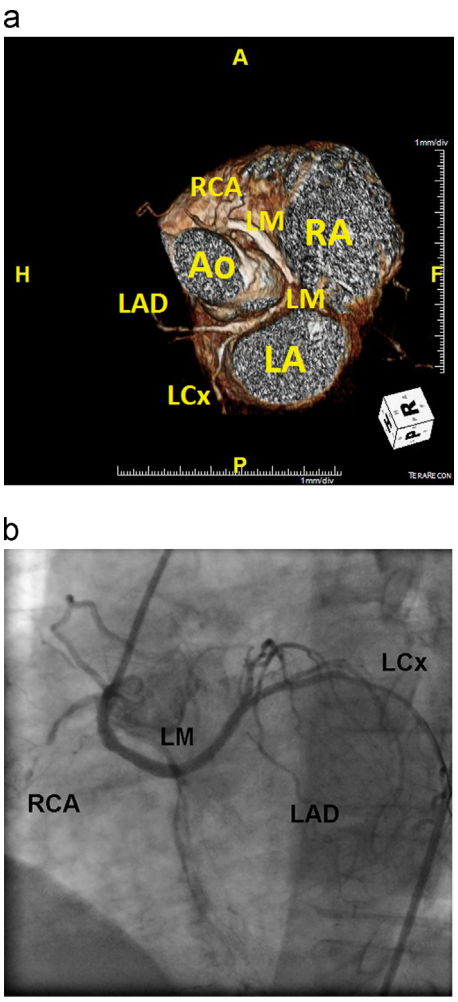

Fig. 5 – The left main coronary artery (LM) arising from the right coronary sinus with retroaortic course. An 80-year-old lady with NSTEMI, chronic occlusion of the right coronary artery (RCA), a tight stenosis of the left main coronary artery, before and after PCI with drug eluting stent implantation. a – MS-CT volume-rendered 3-D image; selective coronarography: b – after PCI with stent implantation: left anterior oblique 45° view.

ridge and further compression of the artery due to dilatation of the aorta during extreme exertion with subsequent myocardial ischemia and sudden cardiac death [1,11].

According to the latest IVUS (intravascular ultrasound) study by Angelini et al. [2,12,13] almost all arteries with an interarterial course have a variable extent of intramural course (the artery is enclosed in the aortic wall, i.e. intussusception). This intramural course represents the basic mechanism for the development of obstruction.

The following factors contribute to this mechanism:

1) Coronary hypoplasia – according to IVUS examination it was demonstrated that an intramural proximal segment of an anomalous artery has a smaller circumference than the distal extramural segment. The degree of impairment is expressed by the hypoplasia index – the ratio of the diameter of the proximal hypoplastic segment to the distal reference segment, which ranges from 0.3 to 0.7 [2,12,13].
2) Lateral compression – the intramural segment does not have a round, but ellipsoid shape. An artery of such diameter has a smaller area than an artery with a round diameter of the same circumference. The asymmetric index relates to a ratio of the smallest and largest diameter of an artery. The smaller diameter of an artery is further compressed laterally during each systole, which results in further intermittent deterioration of flow. This mechanism is enhanced during exertion, which results in changes of stroke volume, tachycardia and other enlargement of the pulsatility of the ascending aorta with subsequent negative feedback.
3) Length of the lesion – the length of any stenosis is another parameter of its haemodynamic significance. In ACAOS the length of the intramural segment is usually 5–15 mm [2].

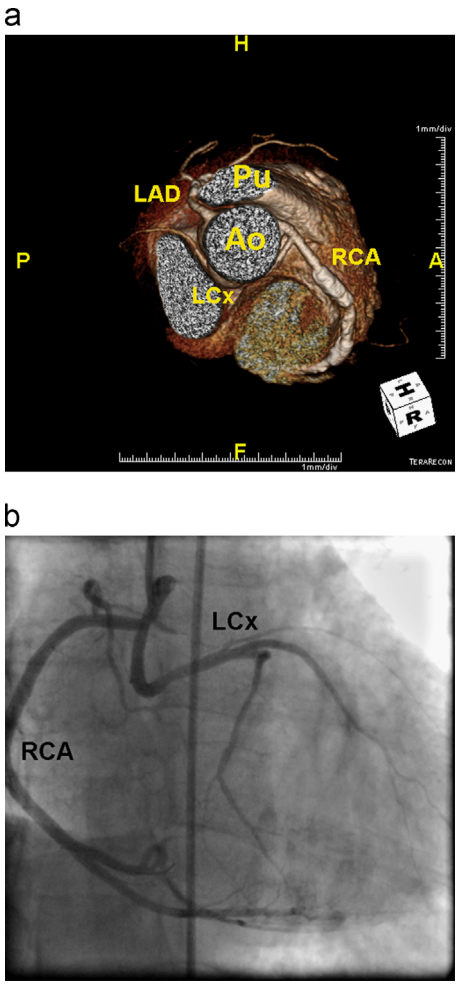

Fig. 6 – The left circumflex artery (LCx) arising from right coronary sinus with retroaortic course, the left anterior descending artery (LAD) arising normally from the left coronary sinus. a – MS-CT volume-rendered 3-D image; selective coronarography: b – right anterior oblique 20° caudal 20° view.

These 3 parameters demonstrate a great individual variability in association with the distensibility of the aortal wall, which is affected by anatomical changes of the wall and changes of aortal pressure [12,13]. Whereas in younger adults ACAOS most often manifests as a sudden cardiac death in association with extreme exertion, in elderly patients the symptoms could include atypical chest pain, shortness of breath, palpitations, syncope, etc. These symptoms often develop gradually in association with the development of hypertension. Sudden cardiac death is not often reported in elderly patients, which is probably related to the aortal wall hardening that develops with increasing age [12,13]. Owing to the rare occurrence of these anomalies, their optimal therapy is unclear. Therapy could include avoidance of extreme physical exertion, beta-blockers therapy, PCI with stent implantation in the case of right coronary anomalous origin or aortocoronary bypass in the case of left coronary anomalous origin, analogically as in the case of the left main stem impairment and possibly other corrective surgical procedures [12,13]. However, unambiguous data confirming the postoperative reduction of risk of sudden cardiac death are missing [4].

Anomalous origin and course of the coronary artery may also cause difficulties in the transcatheter treatment of atherosclerotic lesions.

Anomalous origin of the coronary artery from the pulmonary artery

This is the most serious congenital coronary anomaly with the occurrence in 1 of 300,000 live born children. The anomalous artery could arise from any of the 3 sinuses of the pulmonary valve [9]. The anomalous origin of the left coronary artery from the pulmonary artery (ALCAPA) occurs in 0.008% of cases. The anomalous origin of the right coronary artery (ARCAPA) is less frequent; it occurs in 0.002% of cases [9]. This congenital coronary anomaly could be associated with other congenital heart diseases, in ARCAPA in up to 1/3 and in ALCAPA in 5% of cases, including atrial or ventricular defects, tetralogy of Fallot, double outlet right ventricle, patent arterial ductus, etc. [9]. Clinical manifestation depends on the type of anomaly (ALCAPA or ARCAPA) and the dominance of coronary circulation.

ALCAPA (*Bland–White–Garland syndrome*): The left coronary artery arises from the pulmonary artery, and the right coronary artery normally originates from the aorta. The preserved flow from the pulmonary artery to the LCA is due to the high pulmonary vascular resistance in newborns. The oxygen saturation in blood is thus very low; however, it is sufficient for the supply of myocardium. Shortly after birth, when there is a drop of pulmonary vascular resistance, this perfusion subsides and blood flows from the aorta to the RCA and through collaterals to the LCA and pulmonary artery. Further development of symptoms depends on the extent of collateral network between the RCA and LCA. In case the collaterals are missing completely, sudden cardiac death could occur. Signs of myocardial ischemia due to a steal phenomenon could occur if the collateral flow is developed. Most of these patients are already symptomatic in early childhood with angina pectoris on exertion, acute myocardial infarction or heart failure. Without therapy there is a 90% death rate in children during the 1st year of life. If the collateral supply is sufficient and the patient survives, a left-to-right shunt with pulmonary hypertension and signs of heart failure can develop [9,11].

ARCAPA: It usually has a more favorable course and only rarely results in sudden cardiac death in the case of right coronary dominance [9]. (Fig. 7)

Therapy is based on the surgical replantation of the LCA or RCA origin to the aorta, creation of an intrapulmonary conduit (Takeuchi procedure) or, at a higher age, on the ligature of the artery originating in the pulmonary artery and aortocoronary saphenous vein graft.

Anomalies of course

Myocardial bridging

This represents an intramyocardial course of a normally subepicardial coronary artery due to coverage of the heart

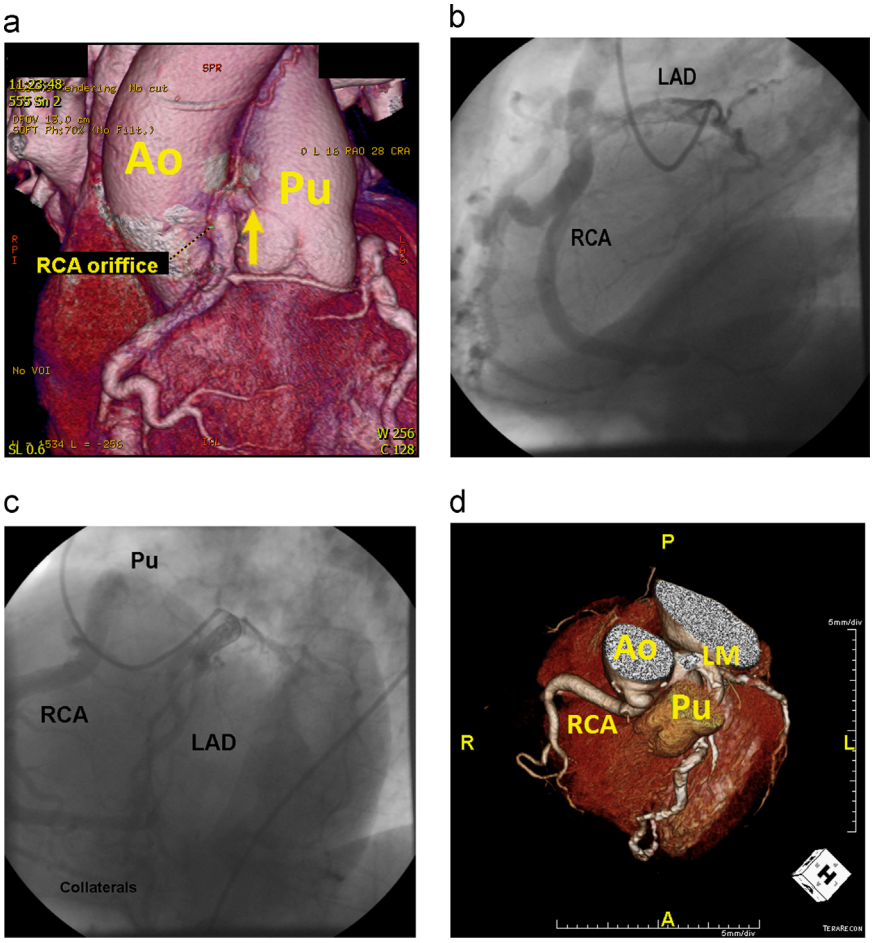

Fig. 7 – Anomalous origin of the right coronary artery from the pulmonary artery (ARCAPA). A 75-year-old patient with effort angina pectoris class CCS 2. Echocardiography has shown hypo- to akinesia of the inferior and lateral walls with reduced ejection fraction of the left ventricle, EF LV 45%. The right coronary origin was not found on selective coronarography, collateral filling from the left coronary artery was detected. MS-CT coronarography demonstrated the anomalous right coronary origin from the pulmonary artery. Re-plantation of the right coronary artery to the aorta was performed. The patient had no postoperative angina pectoris with improved left ventricle ejection fraction. a – MS-CT volume-rendered 3-D images. The right coronary artery (RCA) arises from the pulmonary artery. There is a close relation between Ao and Pu sinuses; selective coronarography: b – Lateral view; c – left anterior oblique 40° cranial 20° view; d – MS-CT volume-rendered 3-D image after the right coronary artery re-implantation to the aorta.

muscle by fascicles. It is most frequently located in the middle segment of the LAD (up to 80% of all cases) [9]. With regard to its incidence in this area it is considered to be a normal variant [9]. A bridge in another localization may be considered anomalous. During coronarography it manifests by a typical "step down – step up" phenomenon that is caused by systolic compression of the artery. MS CT is then able to demonstrate the intramyocardial course of the particular segment. Myocardial bridging is most often completely asymptomatic; it could however cause effort angina pectoris, myocardial infarction or malignant arrhythmia. In this case therapy with betablockers (reduction of heart contractility), stent implantation, myotomy or CABG is indicated [9].

Duplication of arteries

Duplication of the LAD occurs in 0.13–1% of cases [1]. It consists of a short branch in the interventricular sulcus that does not reach the apex and another branch that proceeds more distally, and which arises from the LCA or as an anomalous branch from the RCA.

Anomalies of termination

Coronary artery fistula

Coronary fistula is a communication between the coronary artery and some of the heart chambers, coronary sinus, superior caval vein and pulmonary artery. It occurs in 0.1–0.2% of patients [1]. It originates more often from the RCA (60%) than from the LCA (40%), and in 5% of cases the fistula originates from both branches [1]. Coronary artery fistula is usually dilated and tortuous due to increased blood flow. Termination of coronary fistula has a greater haemodynamic and clinical significance than its origin. The most frequent termination sites are the right ventricle (45%), right

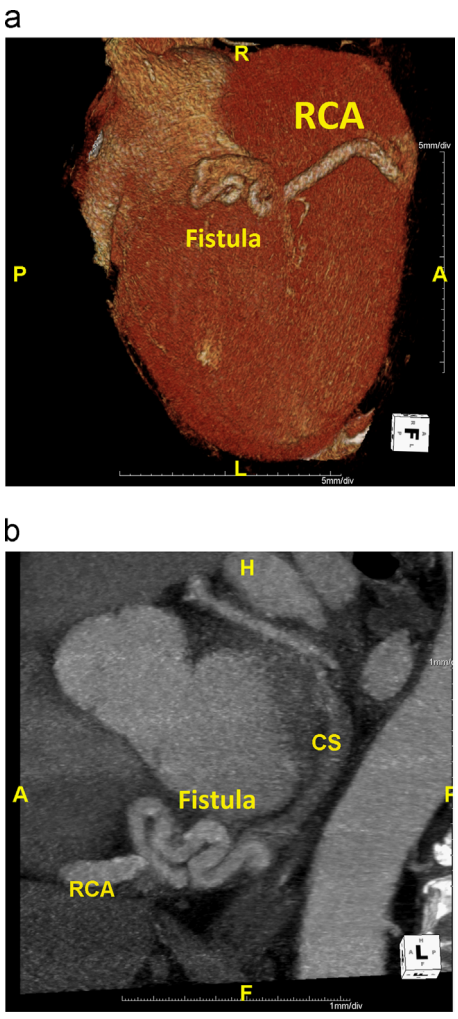

Fig. 8 – Fistula between the right coronary artery (RCA) and coronary sinus (CS) with a haemodynamically significant left-to-right shunt and subsequent development of heart failure. a – MS-CT volume-rendered 3-D image; b – maximum intensity projections (MIP).

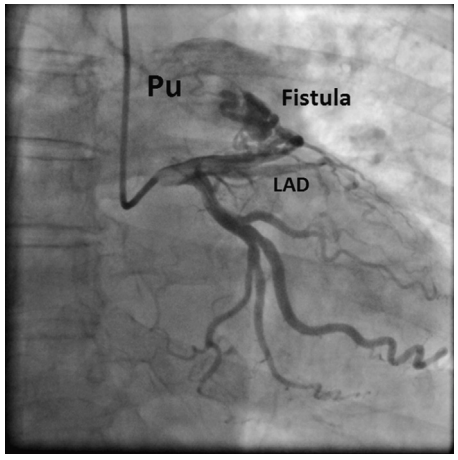

Fig. 9 – Fistula between the left anterior descending artery (LAD) and pulmonary artery (Pu) with a steal syndrome. 9 – Selective coronarography: postero-anterior view.

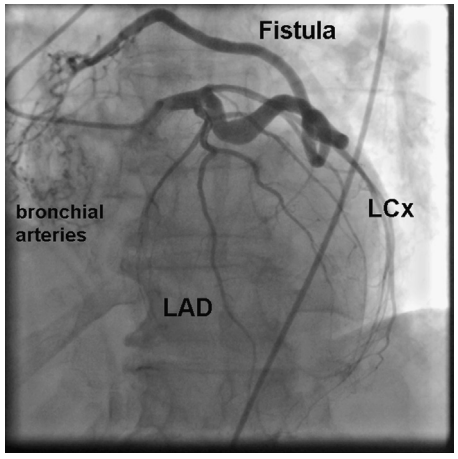

Fig. 10 – Extra-cardiac termination. Fistula between the left circumflex artery (LCx) and bronchial circulation with a steal syndrome. A 75-year-old patient with effort angina pectoris and heart failure class CCS and NYHA 2. Echocardiography has shown akinesis in the inferolateral wall and dysfunction of the left ventricle with EF 35%. 10 – selective coronarography: left anterior oblique 45° cranial 30° view.

atrium (25%) and pulmonary artery (15%). Left sided segments, i.e. the left ventricle or atrium, are involved in only 10% of cases [1]. If the fistula leads into the right sided segments, it results in an extracardiac left-to-right shunt; if the termination is in the left sided segments, it mimics aortic regurgitation [1]. If the fistula is large enough, it could cause myocardial ischemia due to a steal phenomenon in the particular supply area, or in the case of a hemodynamically significant left-to-right shunt it results in heart failure. In addition, an infectious endocarditis in association with a coronary fistula has been reported in ~3% of patients [14]. Rarely large fistulas have been documented to expand aneurysmally and even dissect or rupture [14]. In such cases occlusion of the fistula, embolisation with coils or ligature during a cardiosurgical procedure is indicated [1,3,9]. (Figs. 8 and 9) Approximately 10–30% of patients with a coronary artery fistula also have another congenital cardiovascular anomaly (e.g. Tetralogy of Fallot, patent ductus arteriosus or atrial septal defect) [14]. Fistulas that arise proximally in a coronary artery lead to dilation of only a short segment of the proximal true vessel. These types of fistulas may be the most likely to become grossly dilated and may be at risk for rupture. On the other hand fistulas that arise very distally in the coronary system and are associated with long-segment coronary artery dilation, that persists even after successful closure of distal fistulas, can lead to a late thrombosis of this permanently dilated and tortuous proximal coronary artery. In these cases long term anticoagulation should be considered [14].

Percutaneous catheter closure is applicable to over 90% of patients [14]. It can be achieved with coils or vascular plugs depending on the size of the fistula. The basic technique of

transcatheter closure is to advance a delivery catheter to the most distal portion of the fistula (past any branches that may feed the normal myocardium) and place the occluding device in that location. To check if the closure location is distal enough, first a compliant balloon can be inflated in this location to assess whether ischemia will occur. The coils should be up to 30% larger than the vessel to be occluded at the point of occlusion to avoid inadvertent embolisation of the coil [15]. After selective coronarography a guiding catheter is positioned in the artery. With the help of a 0.035" standard guidewire advanced into the fistula, the guiding catheter may be passed to the point of intended occlusion in cases with a relatively straight course of the feeding artery. In this case, coils can be deployed through this catheter to achieve occlusion. Coils of 0.038" caliber require catheters of 4 Fr or 5 Fr size for their delivery [15]. Positioning such stiff guiding catheters in a distal location in a tortuous fistula may be difficult and hazardous. In these patients controlled-release micro-coils of 0.018" caliber, which can be deployed through a co-axial 3 Fr catheter passed through the guiding catheter, may be preferred. Such catheters can be passed through tortuous arteries into very distal locations over floppy 0.014" coronary guidewires. Controlled-release coils make the procedure more controlled and potentially reversible, because they could be positioned and withdrawn back into the catheter, if the final position is not satisfactory [15]. For large fistulas the use of vascular plugs (e.g. Amplatzer Vascular Plug) or other devices (e.g. Amplatzer PDA) should be considered. The plugs are attached to a delivery wire and can be deployed via 4 Fr or 5 Fr catheters [14,15]. When positioning is optimal, plugs are detached and released. In some patients, it may be easier to enter the fistula by the retrograde approach from the right side of the heart. Or in some large, high flow fistulas, it may be advantageous to first pass a microcatheter and guidewire completely through the fistula. The arterial guidewire can then be snared and exteriorized on the venous side to provide a guidewire rail. This will enable advancement of even a large delivery system and then a vascular plug or coil through the vein, preferably by the retrograde approach. Finally a covered stent may be a suitable device to close some coronary artery fistulas. The origin of the fistula from the normal coronary artery is excluded by placing a covered stent in the main vessel. However, it could be associated with a high rate of re-stenosis. Small residual leaks after either type of procedure appear to be present in ~10% of patients [14,15]. The main complications include inadvertent coil embolisation, which may occur as a result of high flow in the large fistulas or with undersized coils. But in most cases, it is possible to retrieve the devices with goose-neck snares [14,15]. Surgical closure is the preferred approach in patients who are undergoing operative repair of other cardiovascular problems [14].

Coronary arcade

This is a rare instance of communication between the right and left coronary arteries without significant coronary stenosis, which is large enough to be visualized angiographically. Coronary arcade is often a straight connection localized at the level of the crux, in contrast to the tortuous collateral vessels between the patent and the obstructed vessel.

Extracardiac termination

There could be a connection between the coronary and non-coronary arteries (bronchial, internal mammary artery, intercostal artery, etc.). Small connections between bronchial arteries and coronary circulation exist in virtually all patients. These connections could become significant in the case of coronary artery obstruction when blood flows downwards from the bronchial branches to the coronary circulation [1]. (Fig. 10)

Conclusion

Congenital coronary anomalies are relatively rare findings; however, some of these could have significant and even fatal consequences. These disorders should be considered in selected risk population groups. Multi-slice CT coronarography seems to be the most appropriate diagnostic method with regard to the possibility of 3D imaging.

Conflict of interest

We hereby declare that there is no conflict of interest concerning the work published in our paper.

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Ethical statement

We hereby declare that the publication ethics were followed during our paper preparation.

REFERENCES

[1] S.Y. Kim, J.B. Seo, K.H. Do, et al., Coronary artery anomalies: classification and ECG-gated multi-detector row CT findings with angiographic correlation, RadioGraphics 26 (2006) 317–334.

[2] P. Angelini, J.A. Velasco, S. Flamm, Coronary anomalies: incidence, pathophysiology, and clinical relevance, Circulation 105 (2002) 2449–2454.

[3] A.R. Zeina, J. Blinder, D. Sharif, et al., Congenital coronary artery anomalies in adults: non-invasive assessment with multidetector CT, The British Journal of Radiology 82 (2009) 254–261.

[4] P. Angelini, Coronary artery anomalies–current clinical issues: definitions, classification, incidence, clinical relevance, and treatment guidelines, Texas Heart Institute Journal 29 (2002) 271–278.

[5] S.P. Van Camp, C.M. Bloor, F.O. Mueller, et al., Nontraumatic sports death in high school and college athletes, Medicine & Science in Sports & Exercise 27 (1995) 641–647.

[6] B.J. Maron, P.D. Thompson, J.C. Puffer, et al., Cardiovascular preparticipation screening of competitive athletes: a statement for health professionals from the Sudden Death

Committee (Clinical Cardiology) and Congenital Cardiac Defects Committee (Cardiovascular Disease in the Young), American Heart Association, Circulation 94 (1996) 850–856.
[7] R.E. Eckart, S.L. Scoville, C.L. Campbell, et al., Sudden death in young adults: a 25-year review of autopsies in military recruits, Annals of Internal Medicine 141 (2004) 829–834.
[8] U. Drory, Y. Turetz, Y. Hiss, et al., Sudden unexpected death in persons less than 40 years of age, American Journal of Cardiology 68 (1991) 1388–1392.
[9] M. Montaudon, V. Latrabe, X. Iriart, et al., Congenital coronary arteries anomalies: review of the literature and multidetector computed tomography (MDCT) – appearance, Surgical and Radiologic Anatomy 29 (2007) 343–355.
[10] M.A. Greenberg, B.G. Fish, H. Spindola-Franco, Congenital anomalies of the coronary arteries. Classification and significance, Radiologic Clinics of North America 27 (1989) 1127–1146.
[11] P.M. Young, T.C. Gerber, E.E. Williamson, et al., Cardiac imaging: Part 2, normal, variant, and anomalous configurations of the coronary vasculature, American Journal of Roentgenology 197 (2011) 816–826.
[12] P. Angelini, S.D. Flamm, Newer concepts for imaging anomalous aortic origin of the coronary arteries in adults, Catheterization and Cardiovascular Interventions 69 (2007) 942–954.
[13] P. Angelini, Coronary artery anomalies: an entity in search of an identity, Circulation 115 (2007) 1296–1305.
[14] L.A. Latson, Coronary artery fistulas: how to manage them, Catheterization and Cardiovascular Interventions 70 (2007) 110–116.
[15] S.A. Qureshi, Coronary arterial fistulas, Orphanet Journal of Rare Diseases 1 (2006) 51.